ON THE PATHOLOGY OF APPENDICITIS.

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IN SEPTEMBER, 1879, I read a paper on perityphlitis, as it was then called, before the Buffalo Medical Association, calling attention particularly to the operative treatment, then new, as advocated by the late Dr. Gurdon Buck. In discussing the pathology I followed the nomenclature of my distinguished teacher, Professor With, of Copenhagen, who described three forms of appendicitis, namely:

1. Peritonitis appendicularis adhesiva, in which the ulceration in the appendix goes so deep that the peritoneal covering is affected and adhesions are formed.

2. Peritonitis appendicularis localis, characterized by local peritonitis and primary abscess

3. Peritonitis appendicularis universalis, in which we have diffuse peritonitis by perforation into the peritoneal cavity.

The cases belonging to the first division were those with obscure symptoms, local tenderness in the ileo-cæcal region, a little vomiting and general ill-feeling for a few days. I stated that they recovered generally promptly by rest, opium, poultices and avoiding cathartics, but that they frequently relapsed and might then be followed by the more severe or fatal forms.

The second form, peritonitis appendicularis localis, I described as characterized by local abscess, generally and primarily intra-peritoneal, but on account of adhesions in reality extra-peritoneal, and extending downward toward Poupart's ligament, above which they might and ought to be opened by operation as early as possible in order to avoid secondary perforation into the abdominal cavity.

[1]Read before the New York State Medical Society, February, 1891.

The third division, peritonitis appendicularis universalis, I stated could either start as such, if the perforation took place before adhesions had formed, or by secondary rupture of a well-developed abscess. I stated twelve years ago that they almost universally terminated fatally in a few days, that no known treatment was of any avail, but I expressed a belief (page 122, *Buffalo Medical Journal*, 1879) that the time would surely come, when we, in such cases, would open the abdominal cavity and ligate the appendix.

During the last twelve years a great deal has been written about appendicitis, and it is now almost universally acknowledged that it is a distinctly surgical disease, which can be treated by surgical means only, particularly in its more severe forms, where we have either a circumscribed abscess or a diffuse peritonitis.

The three divisions—adhesive, circumscribed, and diffuse peritonitis—are still more or less recognized as the different forms, perhaps under slightly changed names, and the pathology is in most points fully understood and almost beyond discussion.

Bull ("Transactions American Surgical Association") distinguishes between a catarrhal perityphlitis tending toward recovery, but then leaving behind adhesions to the parietal peritoneum, the omentum or the intestines, and a suppurative perityphlitis, which either may be spreading (diffuse peritonitis) or limiting (circumscribed peritonitis), followed by extra-peritoneal abscess.

A catarrhal perityphlitis may go on, he thinks, to a suppurative form too. He considers perityphlitis an inflammation of either cæcum or the appendix with their peritoneal covering on the cellular tissue in the iliac fossa. He thinks it impossible to distinguish between an inflammation of cæcum and the appendix, but, like most writers, believes ulceration leading to perforation more frequent in the appendix. He also mentions that catarrh of the cæcum in which the appendix participates, is of frequent occurrence.

McMurtry, of Kentucky, on the other hand, thinks as most other writers do, that disease about the caput coli in almost every case starts in the appendix, and that inflammation of the cæcum

itself is very rare, although primary perforation of the cæcum may occur without appendicitis. Regnier mentions such a case (1886) and McMurtry another.

My personal opinion is that it may start in either, but with a great predilection for the appendix. Catarrh of the cæcum with dilatation I consider quite frequent, and it is probably followed with catarrh and dilatation of the appendix and its opening into the cæcum, "Gerlach's valve,"so that the contents of the bowels enter with greater ease. The appendix has a relatively large absorbent surface so that the fluid is absorbed while the solid parts are left and form concrements, which again probably are the mechanical causes of inflammation, ulceration, and relapses.

Krafft (*Volkmann's Klinische Vortræge*, January, 1889) thinks resolution impossible, and believes that there is always a pus focus left which may become encapsulated and give no further symptoms, but which also, on slight provocation, may start the inflammation up again. In 84 cases out of his statistic of 106 cases an autopsy was made and in each case an ileo-cæcal abscess found. He also believes perforation of the cæcum always to be secondary, the abscess perforating into the cæcum instead of elsewhere. That resolution may, and does, take place, is conclusively proved by my countrymen, Dr. Toft, of Copenhagen, who, while prosector to the "Royal Frederiks Hospital," found residua of appendicitis in the form of adhesions in 35 per cent of all post-mortem examinations made. It also shows the frequency of the disease.

Mikulicz, of Königsberg (ANNALS OF SURGERY, October, 1889), distinguishes two forms of perforative peritonitis. The first form, diffuse septic peritonitis, results when a large quantity of intestinal contents suddenly pours into the abdominal cavity through a large perforation. The resulting peritonitis is characterized by sanguino-serous or purulent, thin putrid, fluid exudation, injected peritoneum, at times covered by thin fibrinous exudation. Extensive adhesions are lacking, as the patient dies before they can form.

In the second form, which he calls progressive fibro-purulent peritonitis, the peritoneum is first only affected in the immediate vicinity of the perforation, a fibro-purulent exudation

is formed, which prevents by adhesions the infection of the whole peritoneal cavity. The process may spread, closely followed by new exudations, and incapsulated pus foci are formed between the glued intestines. Mikulicz opened in one case six intra-peritoneal pus-cavities through three incisions, in another three pus cavities through three incisions. The openings were made at different times as the existence of the pus foci became evident.

Why we should find this slow progressive form in some cases, and in the majority of cases a violent septic and rapidly fatal peritonitis, is not clear. Less stress has probably been laid upon intestinal microbes than they deserve. It is probably not so much the concrements or the pus that produce the diffuse septic processes in the peritoneum after perforation as it is the pathogenic bacteria, with which the intestinal contents are teaming. Lewis Smith thinks that atrophy and necrosis of the epithelium take place from pressure of foreign substances, that intestinal microbes thereafter invade the exposed sub-epithelial tissue, causing septic inflammation, which extends through the muscular coat to the sub-peritoneal tissue and to the peritoneum, causing local or diffuse peritonitis according to the rapidity of the process. The inflammation may also cease before reaching the peritoneum, causing gangrene and ulceration of the deeper tissues and causing *either* obliteration of the appendix from cicatricial contraction during healing, *or* else stricture of the appendix, particularly the proximal end, while the distal end remains patulous and may form a retention-cyst. This cyst may again subsequently inflame, perforate, and give peritonitis, but probably not a diffuse septic form, but a fibro-purulent form after Mikulicz. Stimson mentions these strictures too, considering them the result of cicatricial contraction and possibly occasionally from congenital defect.

Treves (Discussion in the British Medical Association, August, 1889) does not believe in a catarrhal form of perityphlitis. The perityphlitis is always, in his opinion, produced by ulceration and the symptoms first occur when the ulcers have extended to the outer wall. Be that as it may, I have no doubt that the concrements are the cause of the ulceration and

the catarrh and dilatation the cause of the concrements.

While the three divisions hold good in a large number of cases, it must not be forgotten that pathologists have arrived at this conclusion by examining old and probably neglected cases of appendicitis. It is first during the last two years that a larger number of early laparotomies have been performed on account of acute appendicitis, and that an opportunity of studying the pathological process in the start has offered itself. I believe that to my friend, Dr. McBurney, of New York, belongs the credit of pointing out the real condition of the pathological process in the appendix and of showing that, in some cases at least, we may find no adhesions, and that the large, swollen, almost or wholly gangrenous, appendix may be found freely movable in the abdominal cavity and ready to burst, and without any limiting adhesions to protect the peritoneal cavity. He draws his conclusions from early operations for appendicitis and has found the most varied conditions, from a mild catarrhal condition of the mucous membrane accompanied by some infiltration and thickening of the submucous tissue, to the state of complete gangrene of the whole organ with more or less extensive peritonitis. In one case, operated during a period of health on account of twelve attacks during one year, the appendix was found rigid, swollen, the mucous membrane mildly inflamed, the other tissues thickened but without any evidence of peritoneal inflammation or adhesion.

In another patient who had had four attacks, the operation, performed during health, revealed the appendix firmly bound by old adhesions to the under surface of the intestinal mesentery and cæcum; the appendix was dark-colored and swollen, and there was evidence of former limiting peritonitis. Both of these cases would probably later have terminated in abscess and peritonitis. In one case the appendix formed a cyst containing ℥j dark-brown pus.

In several it was swollen, discolored, but gangrenous only at one or two points where perforation had occurred, and in these cases one or more fæcal concretions existed either within or just without the appendix.

In other cases the appendix was only moderately diseased, but perforation had occurred and recent adhesions had tied it

to some adjacent part, doubling it upon itself and so enclosing a small collection of pus with or without concretions.

In two cases the appendix was completely gangrenous. In all of these cases a plastic peritonitis of greater or less extent existed, always involving the cæcum and generally the adjacent intestinal coils and abdominal wall. In no case was extra-peritoneal inflammation observed. In most cases pus was found more or less confined by adhesions within a limited area, and in one case absolutely no adhesions of any kind existed, though the appendix was perforated by concretions and very foul pus filled the pelvis and ran freely upwards beside the colon.

I have quoted my friend, Dr. McBurney, so freely, because his contribution to the pathology of appendicitis, dependent as it is on personal observations from a number of early laparotomies for appendicitis, is of far greater importance than those observations made in post-mortem examinations upon old and neglected cases. It shows also our utter inability to judge correctly from the symptoms alone about the extent and severity of the lesion, and that from that reason alone laparotomy is and must be the safest method of treatment.

Stimson (*N. Y. Medical Journal*, October 25, 1890) found the same difference in eight early laparotomies in regard to the position of the appendix and the intensity of the inflammation; and in two of these cases, at least, found no limiting adhesions, although perforation from gangrenous spots had occurred. In only one case a foreign body or fæcal concretion was found, but in all marked inflammation of the mucosa, almost obliterating its structure by studding it with round cells. Total or partial obliteration was found in three cases. The pus was intra-peritoneal, except in one case, in which it was found between the layers of the mesocolon. Concrements were only found in one case.

The question whether a perityphlitic abscess is intra- or extra-peritoneal, has been debated again and again. I see no reason for any disagreement on this point. Both cæcum and the appendix are, according to Bull and others, always completely invested with peritoneum. An abscess starting in the appendix must necessarily in the start be intra-peritoneal

limited by adhesions. If the adhesions are strong and exudations continue to be deposited, so that perforation into the abdominal cavity is prevented, the parietal peritoneum may become perforated and the pus is then in the extra-peritoneal tissue in the iliac fossa, *i. e.*, an extra-peritoneal abscess, and may be opened by an extra-peritoneal incision above Poupart's ligament or perforate somewhere else, as into the rectum, the ischio-rectal fossa, or backwards.

McBurney thinks they are always intra-peritoneal and that it is always necessary to cut the peritoneum in order to open such an abscess. I think he stretches that point and I disagree with him.

In one case I discovered a distinct hour-glass abscess consisting of a larger extra-peritoneal abscess, which I opened, and which with a round opening was connected with a deeper lying abscess, in which I could distinctly feel the appendix and a concrement, which I removed. In another much neglected case to which I was called four weeks after the patient was taken sick, because the physician could find no abscess, although looking for it, I discovered an enormous abscess ready to break through Pettit's triangle between the latissimus dorsi and external oblique muscles. Both were extra-peritoneal abscesses although starting as intra-peritoneal abscesses, and in neither was the peritoneum cut. That they are rare is sufficiently proved by Robert Weir, who in 100 autopsies found diffuse suppurative peritonitis 57 times, circumscribed abscess 35 times (in 13 of which diffuse peritonitis was present too) and extra-peritoneal abscess only in four cases. In each of these four cases there was a large ragged opening, showing that an ordinary necrotic process of the peritoneal wall had made the abscess extra-peritoneal.

Krafft mentions flexion of the hip-joint as characteristic of appendicitis, but I must disagree with him on that point. I never saw flexion, *i. e.*, contraction of the ilio-psoas muscle, in appendicitis, and I see no reason why it should occur. The strong fascia iliaca is between the abscess and the muscle; otherwise the point of perforation of the abscess, if left to itself, would be down on the femur below Poupart's ligament. Contraction of the ilio-psoas muscle, in short, occurs only

when the muscle either is acutely inflamed (acute psoitis) or perforated and infiltrated with pus from a cold abscess depending upon caries of the spine, necrosis of the pelvis, etc. A few words may be said about the statistics of relapses, perforations, and fæcal concretions. A person who has recovered from appendicitis without operation is ever after in danger of a relapse, which may be either mild or the most severe form of perforative peritonitis. Krafft mentions a statistic of 106 cases of which 24, *i. e.*, 23 per cent had had previous attacks, generally one to three years previously, in one case twenty years previously. Treves mentions one case who had 14 attacks, McBurney another with 12 attacks inside one year. In regard to perforations, Matterstock found perforations in 132 out of 146 cases, Fenwick in 113 out of 129 cases, *i. e.*, 90 and 86 per cent. The perforation is usually at the free end but may be circular, and as Krafft says, so to speak amputate the appendix.

In Matterstock's 146 cases fæcal concretions were found 63 times, a foreign body 9 times. In Krafft's 106 cases 36 fæcal concretions and 4 foreign bodies were found. Only small bodies can enter on account of Gerlach's valve. A cherry-pit may enter with difficulty, a plum-stone not at all.

In regard to age, Matterstock found in 72 cases, 2 under 2 years of age; 10 between 2d and 5th year; 25 between 5th and 10th year; 35 between 10th and 15th year, which seems to show that childhood is most predisposed. This does not agree with other authors. Of 14 cases I have seen myself, only 1 was a child, a boy 12 years of age; all the rest occurred in adults, and quite a number in people over 40 years of age. One only was a female, the rest males.